CHRONIC CONSTRICTIVE PERICARDITIS: PARTIAL PERICARDIECTOMY AND EPICARDIOLYSIS IN TWENTY-FOUR CASES*

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CHRONIC CONSTRICTIVE PERICARDITIS is the term most generally accepted to designate an inflammatory lesion of the pericardium and epicardium in which fibrous adhesions often associated with deposits of calcium and occasionally pockets of encapsulated fluid form on and between these coverings of the heart and in which the inflammatory scar contracts around and onto the heart muscle to such a degree that it interferes with the normal diastolic and systolic functions of the heart and causes impairment of circulation.

The circulatory failure produced by this condition develops slowly and insidiously. It is due primarily to progressive mechanical interference with the action of the heart and not to intrinsic cardiac disease, except in cases of long standing in which some associated myocardial atrophy and degeneration result from the constriction. The primary cardiac fault in this condition is physiologic due to the fact that the dense inflammatory scar encasing the heart muscle prevents the heart from attaining its normal diastolic volume and thereby causes increased intraventricular tension which results in an inflow stasis.

It is generally accepted that ventricular output is proportional to diastolic filling. In constrictive pericarditis diastolic filling is less than normal and decreased stroke volume and minute output of the heart result. It is probable that other factors may contribute to the decreased ventricular output to a less but significant degree. One of these factors may be interference with the systolic contraction of the ventricle. This interference is attributable not only to fixation of the scar to the myocardium but also to a progressing degree of myocardial atrophy and degeneration from the limited action as well as from the original infectious process.

The primary infectious process which causes constrictive pericarditis may not be and often is not associated with acute symptoms which indicate that the pericardium has been involved. A considerable interval may intervene between the time of recovery of the patient from the primary acute infectious disease which may have been some pulmonary infection and the occurrence of symptoms indicating that the pericardium has been involved in the infection.

ETIOLOGY

The etiology of this condition is of considerable interest and importance and there is some difference of opinion concerning the type of infecting

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organism. Every effort was made to determine the causative agent in the series of 24 cases on which this paper is based. The clinical history of previous infectious diseases and present clinical examinations were considered carefully for etiologic factors and also microscopic examinations and cultures were made of the tissue removed at operation in all cases. This study definitely proved that the etiologic agent in five cases (21 per cent) was tuberculosis. This type of infection, therefore, should always be considered as a possible cause of the disease.

In 19 (79 per cent) of the cases the type of primary infection was unknown. Eight of these 19 patients gave a history of one or more previous attacks of some pulmonary infection, such as pneumonia or influenza. One gave a history of mumps two weeks preceding the onset of symptoms and one, a history of scarlet fever seven years prior to examination at the Mayo Clinic. Nine did not give a history of any infectious process to which the condition could be attributed. Of the patients who gave a history of previous infectious process, only one presented any indication that the pericardium had been involved at the time of the infection. This patient gave a history of pneumonia at the onset of his illness during which pericardial paracentesis was done, with negative findings.

I do not believe it possible, or advisable, to attempt to draw any definite conclusions as to the cause of the disease from this relatively small group of cases but it is of interest to note that in only one of the cases was there a previous history of rheumatic fever and in this instance the pathologic study of the tissue removed at operation revealed that tuberculosis was the etiologic agent.

Those cases in which the etiologic factor cannot be determined are of particular clinical importance as there is a greater possibility of error in diagnosis when the etiology is obscure.

CLINICAL FEATURES AND DIAGNOSIS

The two most common conditions with which constrictive pericarditis is most likely to be confused clinically, are cirrhosis of the liver and congestive heart failure owing to intrinsic cardiac disease. In most instances, however, the subjective symptoms of constrictive pericarditis are sufficiently characteristic that, when they are correlated with the physical and laboratory findings, a definite diagnosis can be established.

Development of factors which produce the clinical syndrome of constrictive pericarditis is slow but progressive. The amount of blood entering the heart and being pumped into the circulation by the heart per beat and per minute gradually decreases. This decrease results in low blood and pulse pressure, faint heart sounds, tachycardia, paradoxical pulse and auricular fibrillation. The back pressure on the venous circulation results in high venous pressure, dilatation of veins, enlargement of the liver and transudation of fluid into the tissues of the body, particularly into the abdomen and pleural cavities, as well as into the tissues of the extremities and face.

The number and severity of subjective symptoms increase progressively with the course of the disease; the earliest symptoms are general weakness and fatigue. Dyspnea is noted on exertion and usually is relieved by rest, particularly by lying down. Orthopnea is experienced rarely. Digestive disturbances, such as anorexia, epigastric distress and fullness in the abdomen after meals, usually are not noticed until after the liver is enlarged considerably and probably can be attributed to hepatic damage, for these symptoms seem to be more severe in cases in which the tests of hepatic function reveal a considerable degree of retention of dye. Swelling of the abdomen does not occur early in the course of the disease; however, it may be one of the first symptoms noticed by the patient. When ascites develops, it usually becomes progressively more extensive. Edema of the extremities which may extend to the face and neck usually does not occur until after ascites has been present for some time. When venous congestion has advanced to this degree, the entire venous system usually is dilated markedly. This dilatation is particularly noticeable in the veins of the cervical region. The effects of this condition cause increasing disability which gradually becomes total disability unless surgical relief is obtained.

The laboratory findings in chronic constrictive pericarditis give valuable aid in establishing a definite diagnosis. The venous pressure is elevated above normal. The circulation time of the blood is increased. The tests of hepatic function indicate a varying degree of hepatic damage which depends to some extent on the duration of the disease. In the series of 24 cases reported in this paper, bromsulfalein tests of hepatic function revealed some degree of retention of dye in all cases. In two cases retention of dye was Grade 1 (on a grading basis of 1 to 4), in 17, Grade 2, in four, Grade 3, and in one case Grade 4.

Roentgenologic studies of the heart in cases of constrictive pericarditis usually reveal it to be normal or smaller than normal, occasionally slightly enlarged but never markedly enlarged or segmentally dilated as in congestive heart failure from intrinsic cardiac disease and hypertensive heart disease. The presence of calcareous plaques in the pericardium is noted frequently. This is always suggestive of the possibility of a constricting pericardium but calcium does occur in the pericardium without other findings of constrictive pericarditis. I have had under observation two patients who have calcification of the pericardium, for one and two years, respectively. The venous pressure and circulation time of the blood in these two cases were normal. In the usual case of constrictive pericarditis roentgenoscopic examination reveals diminution of cardiac pulsations.

The electrocardiographic findings, although not pathognomonic, are suggestive. In a previous paper published in 1940, Barnes and I[1] made the following statement on electrocardiographic findings: "The electrocardiographic pattern which is most diagnostic is one in which the QRS complexes are of low voltage (below 5 Mm.) and T waves are negative in all the standard leads. If the QRS voltage is low in only one lead that will usually be

observed in lead I. The T waves in the standard leads may be of low voltage though upright, they may be iso-electric or may be inverted in only two leads. The Wolferth precordial lead is characterized by an exaggerated Q wave at the expense of an R wave of diminished amplitude. Similarly the R wave in leads IV R and IV F is disproportionately large as compared to the S wave. The T wave in the Wolferth precordial lead is upright in more than half the cases and it frequently arises from an R–T segment that is slightly elevated. The T wave in lead IV R is negative in about half the cases and it arises frequently from an S–T segment that is slightly depressed. The electrocardiograms change very little after operation in the cases in which the operation is not successful in relieving inflow stasis. In cases in which inflow stasis is relieved by operation the electrocardiogram tends to return to normal after operation though some trace of the original electrocardiographic abnormalities may persist for a long time in spite of the fact that normal cardiac function has been restored." The most important clinical features in my series of 24 cases are shown in Table I. These indicate that the most important clinical manifestations of constrictive pericarditis are increased venous pressure, increased circulation time, enlargement of the liver with impaired function, ascites, faint heart sound and diminished force of pulsations, low blood and pulse pressure and the heart of normal size or not appreciably enlarged.

Inasmuch as chronic constrictive pericarditis is essentially a mechanical condition interfering with the function of the heart, it can only be relieved by surgical removal of this fibrous encasement of the heart muscle.

It is important that the diagnosis be made early and operative treatment instituted as soon as the diagnosis is established because the longer surgical intervention is delayed the more extensive the myocardial degeneration will become and the more destructive the effects of the disease will become on other body tissues, especially the liver. The greater the degree of myocardial atrophy and degeneration the greater the hazard of operation and the less satisfactory the ultimate operative results.

SURGICAL TREATMENT

My experience in the surgical treatment of constrictive pericarditis consists of this series of 24 cases in which partial pericardiectomy and epicardiectomy with epicardiolysis were performed. I have used the term "epicardiolysis" to designate the separation of the innermost layer of the pericardium from the heart muscle. This procedure is used in addition to resecting a portion of pericardium and epicardium. Epicardiolysis is an important and essential part of the operative procedure. Nine of these cases were reported by Barnes and me in 1940.

Preoperative Treatment.—The preoperative preparation of patients who have been selected for surgical treatment is directed at removing the effects of the circulatory failure owing to the constriction of the heart and at improving the function of the structures involved.

The intake of fluid should be limited to from 1,200 to 1,500 cc. in 24 hours. Patients should receive a diet high in protein and low in salt. Daily administration of vitamins may be of value in improving the general resistance of the patient. When tests of liver function indicate considerable damage, vitamin B-complex and glucose administered intravenously, or by mouth, may be helpful in improving the function of the liver.

The body cavities and tissues should be relieved of as much excess fluid as possible. When large amounts of fluid are present in the abdominal and pleural cavities, the fluid should be removed by aspiration and periodic use of diuretics should be utilized to relieve edema of the tissue and keep it at a minimum.

The value of preoperative administration of digitalis is questionable. In the first cases digitalis was given before operation but its use before operation did not produce appreciably satisfactory results and has been discontinued except in some cases of auricular fibrillation. I prefer not to have the patient receive digitalis before operation unless its use is definitely indicated because its stimulating effect on the heart muscle may result in a false impression of the ability of the heart to withstand the surgical procedure. I believe that if digitalis is not used preoperatively, a better evaluation can be obtained of the amount of irritation the heart muscle can safely withstand during the operative procedure.

The level of blood urea, plasma chlorides and serum protein and the carbon dioxide combining power of plasma are determined routinely before operation to see that they are within normal limits as well as to have a control for the postoperative determinations. The type of the patient's blood is determined and blood of similar type is held available for intravenous administration at the time of operation if it should become necessary to utilize it because of loss of blood or severe decrease in blood pressure. Blood or other fluids, however, should not be administered intravenously during operation unless their use is indicated specifically.

Preliminary Medication.—Preliminary medication is begun the night before operation and consists of 1.5 grains (0.1 Gm.) of pentobarbital sodium taken orally. Two hours before the time of operation another dose of 1.5 grains (0.1 Gm.) of pentobarbital sodium is administered by mouth. This dose often is repeated an hour before operation. Thirty to 45 minutes before the time of operation $\frac{1}{6}$ grain (0.01 Gm.) of morphine sulfate and $\frac{1}{150}$ grain (0.00043 Gm.) of atropine sulfate are administered hypodermically. Morphine should be used with caution and in sufficiently small amounts so that it will not interfere with respiratory function. Amounts vary with the age and condition of the patient. I believe that it is advisable to omit its use for children less than 15 years of age.

Anesthesia.—General anesthesia is advisable because the operation may require considerable time. This time factor is tiring for the patient and if general anesthesia were not employed, the nervous strain would be increased considerably and would interfere with cardiac function. Furthermore, if

respiratory difficulties arise, it is advisable to have the patient under complete control. The anesthetic agent is administered by means of a positive pressure apparatus. In my early cases this administration was effected through a closed mask but I now use an intratracheal tube routinely. This change was not effected because of any serious trouble with a closed mask but I feel that the equation of safety is greater if the intratracheal tube is used. It may be, and probably is, unnecessary in some cases, but I do not believe that its routine use produces any harmful effects and if its use becomes urgent, it may be difficult to introduce the tube and serious consequences may result before it can be introduced. Use of the intratracheal tube makes it possible to maintain adequate oxygenation at all times and under all circumstances because it insures an adequate air-way and mucus or secretions can be easily aspirated through the tube. It is also an added protection against respiratory dysfunction arising from collapse of the lung if the pleura should be inadvertently opened on one or both sides.

I prefer cyclopropane as the anesthetic agent. In my earlier cases cyclopropane was used routinely. At present I use cyclopropane in combination with ether. The reason for this modification has been to lessen the occurrence of cardiac arrhythmia which may be associated with cyclopropane anesthesia.

Operative Procedure.—The patient is placed in a straight dorsal position, on the operating table.

A U-shaped skin incision is made to approach the pericardium extrapleurally through the left anterior portion of the thoracic wall. This incision begins at about the anterior axillary line at the level of the sixth rib; it extends across the thorax to the sternum; the apex of the curve is in the midportion of the sternum and the upper end of the incision extends as high as the third rib on the anterior axillary line.

The pectoralis major muscle is dissected and is reflected with the skin flap. The costal cartilages of the sixth, fifth, fourth and in some instances of the third ribs on the left side are resected subchondrally and subperiosteally from the sternum together with 3 cm. of the corresponding ribs. An intercostal muscle flap is then cut from the left border of the sternum and the intercostal vessels are cut and ligated. The internal mammary vessels are ligated in the upper and lower limits of the wound in the thoracic wall, usually beneath the perichondrium of the third and sixth costal cartilages. After the muscle flap is mobilized, any existing pericardial attachments to the sternum are separated and a portion of the left half of the sternum is resected with the rongeur. In some instances it is not necessary to remove any of the sternum beyond the detachments of the cartilages.

The left pleura may be dissected from the pericardium at this time. The pleura, however, is often adherent, particularly when the pericardium is calcified, and openings may be made inadvertently into the pleura during its separation. Any openings, thus occurring in the pleura, are immediately closed with catgut. In cases in which only a little pericardial space is free of pleural attachments and in which the pleura is thin and adherent to the

outer wall of the pericardium, it is not advisable to make a wide separation of the pleura from the pericardium until after dissection of the pericardium from the myocardium has been made because of the danger of opening the pleural cavity at this early stage of the operative procedure. Inadvertent opening of the pleural cavity at this time, however, may result in pulmonary

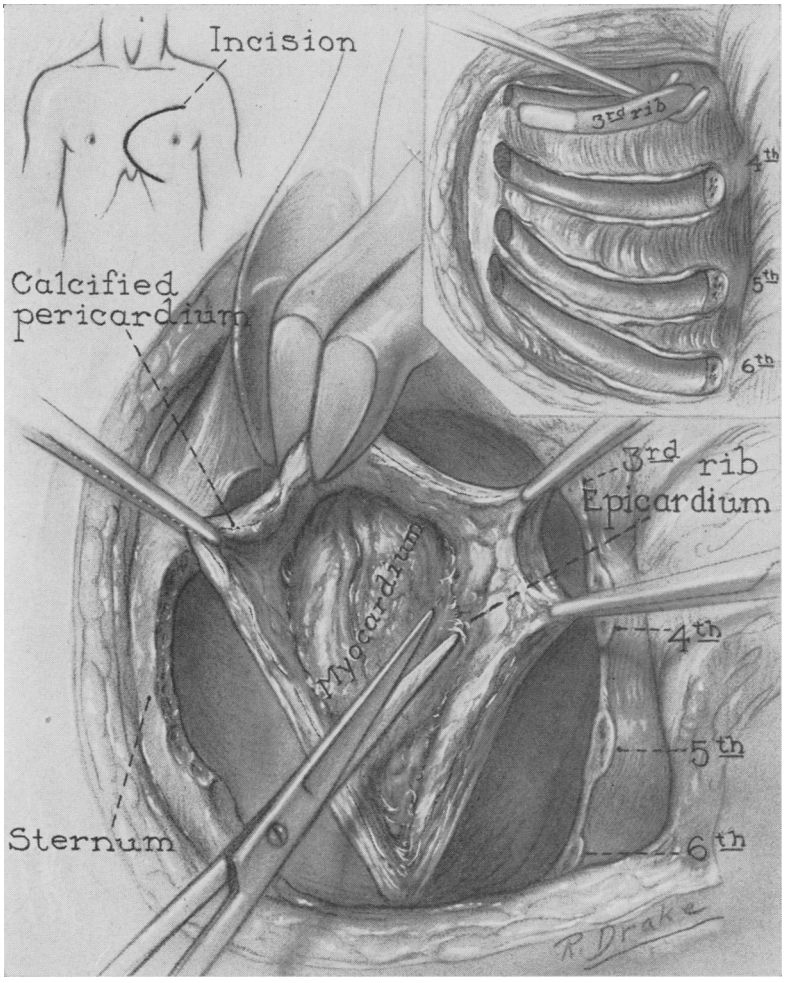

FIG. 1.—Sharp dissection of the constricting epicardium (epicardiolysis) and pericardium from the myocardium. Bone cutters were required for resection of the scar because of the thickness of the calcification. *Left Inset:* U-shaped precordial incision. *Right Inset:* Approach to the pericardium through the wall of the thorax. Resection of the cartilages and 2 cm. of the third, fourth, fifth and sixth ribs on the left side (resection proximal half of sternum).

collapse and respiratory difficulty which may interfere seriously with the patient's progress throughout the rest of the operation. Usually sufficient exposure of the heart can be obtained to complete the dissection of the adherent pericardium from the myocardium without separating the attachment of the left pleura to the pericardium until resection of the pericardium is about to be performed.

The initial incision into the pericardium is important. An attempt is made to make the incision in a region which is removed from the underlying coronary vessels. The site usually selected is close to the sternum or well over the left ventricle in order to avoid injury to the anterior descending portions of the left coronary vessels. The site of election depends to some extent on the type of scar and accessibility of the myocardium. Great care should be exercised to ascertain the actual point of fusion of the pericardial scar and the myocardium before removal of the epicardium is started. The separation of the epicardium from the myocardium is very important and is done in addition to the resection of a portion of the epicardium and pericardium and extends over a much wider area of heart muscle than the area from which the scar is resected (Fig. 1). The site of fusion of the epicardium and myocardium often varies greatly, particularly when deposits of calcium are present. In some instances the calcified tissue may extend into the heart muscle and great care is required in removal to avoid serious injury to the heart muscle. During removal of this scar the deposits of calcium should be left attached to the reflected pericardium. In some regions, particularly around the deposits of calcium, the fusion plane is not uniform and the dissection must be carried around the deposit in slightly different planes before it can be removed by sharp dissection. When the calcium invades the heart muscle, no attempt should be made to remove it as the tissue involved may extend entirely through the heart muscle. In instances in which the scar tissue has extended into the heart muscle, small segments of scar should be left attached to the myocardium because of the risk of damaging the muscle or the coronary vessels in their removal. As mentioned previously, in many instances the scar presents irregular lines of cleavage. It often extends into the myocardium at different levels. In these instances it is best not to follow one line of cleavage to the point of injury to the myocardium but to shift into another line of cleavage and then separate the intervening bands to avoid myocardial injury.

An attempt is always made to free the left ventricle first because better function of this chamber of the heart should be established to provide better disposal of the blood before freeing the right side of the heart. In many instances, however, I have separated the greater portion of the right ventricle first without harmful effects.

Opinions differ concerning the amount of pericardial scar that it is necessary to remove as well as concerning the amount of scar to be separated from the heart muscle and from the orifice of the inferior vena cava. I believe it is advisable to separate as much of the pericardial scar as possible from the ventricles, the right auricle and orifice of the inferior vena cava and it is of particular importance to separate the attachment of the right ventricle to the diaphragm. It is of equal importance to separate the apex of the heart and I believe that this should be done early in the operation if possible. Separation of the heart muscle from its fixed attachments to the diaphragm is one of the most important considerations in reestablishing the action of the

heart. Improvement in the function of the heart often is noted immediately after the apex and right ventricle have been separated from their fixed attachment to the diaphragm.

After the scar has been separated from the heart muscle, the amount of pericardium to be resected depends on the character of the scar and the position and type of fixation of the pleural attachments to the outer wall of

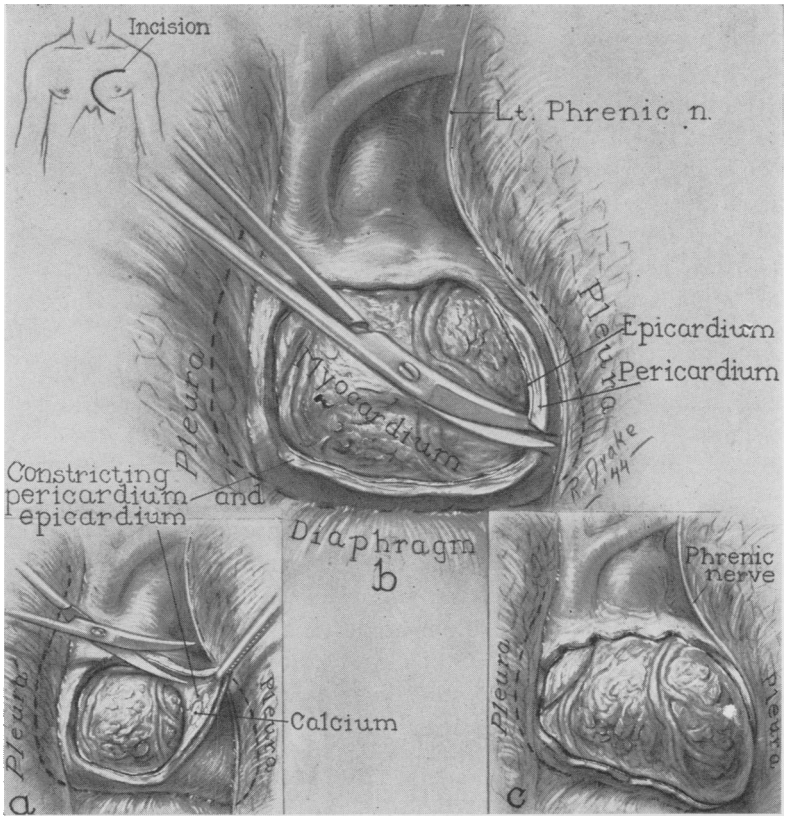

FIG. 2.—Resection of, and marginal incisions in, the pericardium and epicardium after separation from the myocardium. *Inset a:* Beginning resection of the pericardial scar from the anterior and larger surface of the myocardium. *b and Inset c:* After resection of the pericardial scar, multiple incisions are made into the cut margin of the remaining epicardium and pericardium in order to release the heart more fully, particularly at the apex.

the pericardium. I believe it is advisable to resect as much of the anterior portion of the pericardium as possible without injuring the pleura. By careful dissection the pleural attachments to the pericardium can be separated to approximately the position of the left phrenic nerve and the entire anterior portion of the thickened pericardium extending beneath the sternum is resected. If the attachments of the pleura extend high on the pericardium as they often do when calcium is present, it may be impossible to resect an adequate amount of pericardium without injuring the pleura. In these

cases linear incisions into the remaining cut edges of the pericardium will produce a much wider opening of the pericardium (Fig. 2).

The importance of separating and removing sufficient scar to release the heart cannot be overestimated because, if an adequate amount is not removed, cardiac action will continue to be impaired. This not only will eliminate the possibility of ultimate recovery from the disease but will interfere with the immediate recovery from the operation.

In the course of removal of the pericardial tissue the pulse rate often becomes rapid and irregular owing to the external stimulus to the heart muscle. When the pulse becomes irregular, it is important to cease the operation temporarily. The utilization of frequent rest periods and applications of sponges moistened with warm physiologic solution of sodium chloride will permit the heart to regain or at least partially regain its former rhythm.

The upper half of the wound down to the bed of the cartilage of the fifth rib is closed in two layers. Above the cartilage of the fifth rib the intercostal muscles are sutured to the sternum. The perichondrium of the cartilages of the fifth and sixth ribs is excised completely. From the cartilage of the fifth rib to the lower angle of the wound the subcutaneous tissue and skin only are closed over the heart. Soft rubber tissue drains are placed in the lowermost angle of the wound between the heart and the diaphragm. These drains permit the immediate removal of the serous exudate from the precordial space. The drains are removed from 36 to 48 hours after operation.

A varying amount of serous exudate always follows removal of the adhesions, even though great care is taken to prevent trauma to the tissues as well as to attain accurate hemostasis. If this fluid is permitted to accumulate in the precordial space, it may interfere with the function of the heart which is already laboring under increased stress from the operation. If it is not permitted to drain externally, the fluid may rupture into the pleural space and interfere with respiratory function. When granular deposits of calcium are scattered throughout the pericardium, it is particularly advisable not to have this material drain into the pleural cavity because of the risk of pleural irritation and empyema.

POSTOPERATIVE CARE

Immediately after operation all patients are placed in an oxygen tent. The time during which administration of oxygen is continued depends on their progress. This is one of the most important aspects of postoperative treatment, for the oxygen not only prevents cyanosis but aids in relieving stress on the heart. Other postoperative measures are directed toward removal of body fluids and aiding the function of the heart and liver. These measures are similar to those utilized preoperatively for similar purposes. Limitation of the intake of fluid is important; from 1,200 to 1,500 cc. of fluid is administered in 24 hours by proctoclysis or by mouth as soon as nausea subsides. The intravenous administration of fluids is avoided if possible. In instances

in which the urinary output is reduced, 10 to 20 per cent solution of glucose may be administered intravenously. This not only aids the urinary output but may benefit the function of the liver. Vitamin B also is given.

TABLE I

CLINICAL DATA IN TWENTY-FOUR CASES OF CONSTRICTIVE PERICARDITIS

Duration of Symptoms: 3 months to 15 years

Sex	Patients	Age of Patients, Yrs.	Patients
Males	18	1–19	8
Females	6	20–29	6
	—	30–39	7
Total	24	40–49	3
	Clinical Features		
Ascites	19	Increased venous pressure (190 to 360 Mm. of water)	24
Edema of legs	16		
Edema of face	6	Increased circulation time (highest 90 sec. with decholin)	16
Pleural effusion	7		
Dyspnea	16	Low blood and pulse pressure	20
Orthopnea	2	Diminished pulsations	24
Cyanosis	3	Auricular fibrillation	4
Weakness	12	Enlarged liver (dye retention)	24
Dilated veins in neck	10	Heart small or normal	18
Gastric distress	8	Heart slightly enlarged	6

TABLE II

NINE CASES OF CHRONIC CONSTRICTIVE PERICARDITIS IN WHICH CURE RESULTED FROM PERICARDIECTOMY

Case	Age, Yrs. and Sex	Duration: Symptoms	Duration: Ascites	Venous Pressure, Mm. of Water	Etiology (Previous Infections)	Fibrous Adhesions	Results and Time after Operation
1	27 M	8 yrs.	8 mos.	310	Unknown (pneumonia, 15 yrs. ago)	x Calcium	Cured, 6 yrs.
2	38 M	9 yrs.	5 yrs.	260	Unknown (influenza, 11 yrs. ago)	x Calcium	Cured, 5 yrs. 10 mos.
3	37 F	1 yr.	0	235	Unknown (none)	x Calcium	Cured, 5 yrs. 1 mo.
4	21 M	10 mos.	10 mos.	360	Tuberculosis	x	Cured, 3 yrs. 9 mos.
5	11 M	4 mos.	4 mos.	280	Unknown (none)	x Calcium	Cured, 2 yrs. 10 mos.
6	21 M	6 mos.	6 mos.	284	Unknown (empyema, 7 yrs. ago)	x Calcium	Cured, 2 yrs. 10 mos.
7	37 F	2 yrs.	1.5 yrs.	238	Unknown (none)	x	Cured, 1 yr. 10 mos.
8	14 F	2 yrs.	2 yrs.	315	Unknown (none)	x Calcium	Cured, 1 yr. 7 mos.
9	33 M	3 mos.	2 wks.	282	Unknown (influenza, 1 yr. ago)	x Calcium	Cured, 2 yrs. 10 mos. Died: Auto accident

RESULTS FOLLOWING OPERATION IN TWENTY-FOUR CASES

The rapidity and completeness with which recovery occurs after operation for constrictive pericarditis depends on many factors, the most important of which are the amount of myocardial atrophy and degeneration, the amount of hepatic damage, and the thoroughness with which the constricting scar has been removed from the impaired heart. Improvement may be noticed soon after the operation but it proceeds slowly and progressively. Ultimate

complete recovery may not occur for many months. This is somewhat dependent on the duration of the disease before operation. One patient (Case 1) did not obtain complete recovery for nearly two years following the operation.

Of the 24 patients operated upon, 18 recovered from the operation and six died in the hospital following operation. The operative mortality was 25 per cent.

Tables II, III and IV give some of the clinical data and the results of operation in this series of 24 cases. This series includes nine cases (Cases 1, 2, 3, 9, 15, 16, 19, 21 and 22) which were reported in 1940.

TABLE III

NINE CASES OF CHRONIC CONSTRICTIVE PERICARDITIS IN WHICH IMPROVEMENT RESULTED FROM PERICARDIECTOMY

Case	Age, Yrs. and Sex	Duration		Venous Pressure, Mm. of Water	Etiology (Previous Infections)	Fibrous Adhesions	Results, Time after Operation
		Symptoms	Ascites				
10	34 F	2.3 yrs.	2 yrs.	225	Unknown (none)	x	Marked improvement, 2 yrs.
11	34 M	2.5 yrs.	2 yrs.	235	Unknown (influenza, 15 yrs. ago)	x	Marked improvement, 1 yr. 11 mos.
12	46 M	3 mos.	0	235	Tuberculosis	x	Moderate improvement, 1 yr., 1 mo.
13	16 M	2 yrs.	0	190	Unknown (none)	x Calcium	Progressing improvement, 5 mos.
14	29 F	9 mos.	4 mos.	204	Unknown (influenza, 1 yr. ago)	Encap. fluid; calcium	Progressing improvement, 3 mos.
15*	43 M	15 yrs.	2.5 yrs.	315	Unknown (empyema, 15 yrs. ago)	x Calcium	Improvement (2 operations), 6 yrs. 6 mos.
16	17 M	3 yrs.	2 yrs.	340	Tuberculosis	x	Improved, 2 yrs. 3 mos. Later died of tuberculous peritonitis
17	15 M	4 mos.	4 mos.	255	Unknown (scarlet fever, 7 yrs. ago)	x Calcium	Improved, 7 mos. Later died of pneumonia
18	17 M	6 mos.	3 mos.	350	Unknown (influenza, 1 yr. ago)	Encap. fluid	Improved, 2 yrs. 1 mo. Later died of progressive cardiac failure

* Pneumonia at onset of illness; pericardial paracentesis gave negative results.

Tables II and III give the data on the 18 patients who recovered from operation and include data on four patients who have died subsequently. Table IV gives the data on six patients who died in the hospital following the operation.

Of the 18 patients who recovered from operation, nine (Cases 1, 2, 3, 4, 5, 6, 7, 8 and 9) were cured. By cured, or cure, I mean that they have been able to resume their normal activities with reasonable restrictions. Operation was performed in these nine cases from one year and seven months to six years before this study was made (Table II). One of these patients (Case 9) has died since operation from another cause. After making an excellent recovery and being able to resume his work as a farmer, he was killed in an automobile accident two years and ten months following the

operation. The result in one of these cases (Case 4) was especially gratifying as the etiologic agent in this case was tuberculosis. This diagnosis was proved by microscopic examination of the pericardium removed at operation. This patient is able to carry on his usual activities, and it is now three years and nine months after the operation. Of the five patients who had tuberculosis (Cases 4, 12, 16, 19 and 20), this is the only patient who obtained a result

TABLE IV

DEATHS FOLLOWING PERICARDIECTOMY FOR CHRONIC CONSTRICTIVE PERICARDITIS

Case	Age, Yrs. and Sex	Duration of Symptoms	Etiology (Previous Infections)	Constricting Lesion of Pericardium	Results
19	27 M	1.5 yrs.	Tuberculosis*	Fibrous effusion, (subacute)	Died 29th day; left tuberculous empyema; multiple pulmonary infarcts.
20	26 M	4 yrs.	Tuberculosis	Fibrous	Died 13th day; left tuberculous empyema with bronchopneumonia and pulmonary tuberculosis; cardiac failure.
21	12 M	5 yrs.	Unknown (none)	Fibrous and calcium	Died 4th day; cardiac failure; myocardial degeneration; liver damage; (previous Talma-Morison operation).
22	41 M	4 mos.	Unknown (none)	Fibrous and calcium	Died 3rd day; cardiac failure; bronchopneumonia; myocardial degeneration; calcium through wall of ventricle.
23	16 F	2 yrs.	Unknown (mumps, 2 yrs. before onset)	Fibrous and calcium	Died 3rd day; multiple bilateral pulmonary embolism.
24	12 M	2 mos.	Unknown (none)	Fibrous	Died at operation; cardiac failure.

* Previous history of rheumatic fever.

that could be considered as a cure. In Case 12 the improvement was moderate and in Case 16 the patient died two years and three months after operation from tuberculous peritonitis. The remaining two patients (Cases 19 and 20) died in the hospital after the operation.

Of the remaining nine patients who recovered from operation (Table III), two patients (Cases 10 and 11) have shown marked improvement two years and one year and eleven months, respectively, following operation. One of these patients had received treatment for cirrhosis of the liver for two years. Neither of these two patients has been examined in the past nine months and, although I believe from recent letters of inquiry that they are approximately cured for they are both back to slightly restricted former work, I have placed them in the markedly improved group until further examination. In Case 12 a subacute type of tuberculosis with fibrous adhesions and considerable myocardial damage was present. The patient has been moderately active but is unable to follow any regular occupation one year and one month after operation. His condition is not more than moderately improved and I believe it is questionable whether it will greatly improve in the future. In Cases 13 and 14 sufficient time has not elapsed since the operation to determine the end-result. At present, three and five months after operation, these patients are showing progressive improvement and I believe from their present progress that the ultimate result will approximate a cure.

In Case 15, the disease was of long standing and there was a marked degree of myocardial and hepatic damage. This is the only patient in my series who has had two operative procedures. It is now six years and six months from the first operation and five years following the second operation, and his condition cannot be considered more than improved. While he is now able to carry on moderate activities, he continues to have a moderate degree of ascites. After this length of time, I do not believe his condition will ever improve to approximate a cure.

The remaining three patients (Cases 16, 17 and 18) made satisfactory recoveries from the operation but have died subsequently, two from continuation of the disease and one from other causes. In Case 16, the etiologic agent was tuberculosis. The patient made a moderate improvement following the operation for a period of two years and then his condition gradually declined and he died of tuberculous peritonitis two years and three months following the operation. In Case 17 improvement occurred for seven months after operation at which time an influenzal type of pneumonia developed and caused death. This boy, age 15, although he had a long-standing disease and considerable myocardial damage, would ultimately have had a satisfactory result from the operation, I believe, if pneumonia had not developed. In Case 18 the patient's condition was not satisfactory before operation because of marked hepatic damage, Grade 3, and it was impossible to reduce the amount of fluid in his abdomen or pleural cavity. At the time of operation there was a marked degree of myocardial degeneration and considerable encapsulated fluid. His condition improved only slightly after operation. He became gradually worse and died two years and one month following operation from progressive cardiac failure.

Six patients (Cases 19, 20, 21, 22, 23 and 24; Table IV) died in the hospital after operation. One patient (Case 19) died on the twenty-ninth day after operation of left pulmonary tuberculous empyema and multiple pulmonary infarcts. This patient had subacute tuberculous pericarditis, with effusion. His convalescence was satisfactory until the seventh day when the respiratory rate increased and fever and signs of pleural effusion developed. His condition gradually became worse. He became irrational and died on the twenty-ninth day from multiple pulmonary infarctions and tuberculous empyema. Another patient (Case 20) had extensive tuberculous pericarditis, myocardial damage and pulmonary tuberculosis. The essential cause of his death 13 days after operation was cardiac failure and left tuberculous empyema with congestive bronchopneumonia. In Case 21, the patient died on the fourth day after operation from cardiac failure as a result of myocardial degeneration. This patient had had a Talma-Morison operation. In Case 22, the patient died on the third day after operation from cardiac failure and bilateral bronchopneumonia. Examination of the heart, in this case, revealed a large plaque of calcium which extended throughout the entire wall of the ventricle and presented into the ventricular chamber of the heart. In Case 23, the patient was a frail girl, age 15, who weighed 75 pounds (34 Kg.) when

she was first admitted to the Mayo Clinic in September, 1943. At that time she was extremely ill and her temperature was elevated daily. It seemed questionable whether surgical treatment would be advisable. However, her condition began to improve in about two months and after a period of four months she was free of fever and had gained five pounds (2.3 Kg.) so that operation seemed justified. The operation was without incident, but she died on the third postoperative day of multiple pulmonary embolism. This patient had had digitalis prior to her operation.

In Case 24, death occurred suddenly at the time of operation from cardiac failure. This is the only immediate surgical death in this series. There were no technical complications during the operation. The heart failure occurred after the pericardium had been completely separated but before it had been resected. The heart suddenly stopped during the period of rest when hot sponges were being applied over the pericardium and heart muscle. The patient was frail and had lost considerable weight but after being under preoperative treatment for a month and a half his condition improved moderately and it was felt that surgical treatment was justified. At necropsy the only finding was that of a marked degree of atrophy of the myocardium.

REPORT OF CASE 1

The patient, a man, age 27, was admitted to the Mayo Clinic on April 11, 1938. No definite etiologic factor could be found for his condition. He had had three attacks of pneumonia 15, 8 and 6 years prior to his admission. He had not been well since the attack of pneumonia eight years prior to his admission at which time a deposit of calcium was found around the pericardium on roentgenologic examination. His condition became gradually worse until eight months prior to his admission when ascites and edema of the legs developed. Since that time dyspnea, weakness and fatigue had been severe. He also complained of considerable gastric distress and anorexia.

At the time of his admission abdominal paracentesis was required every six to eight days. The liver was greatly enlarged and a test of liver function revealed retention of dye, Grade 3. The veins of his neck were distended. The venous pressure was 310 Mm. of water. The circulation time, determined by means of the intravenous injection of sodium dehydrocholate, from the arm to the tongue was 43 seconds. Systolic blood pressure was 108 Mm. of mercury and the diastolic was 80 Mm. of mercury. The electrocardiogram revealed a cardiac rate of 90 beats per minute, auricular fibrillation, notched QRS in lead I, slurred QRS in leads II and III, and questionably inverted T waves in standard lead IV. The roentgenograms taken on admission revealed enlargement of the heart shadow with extensive calcification of the pericardium (Fig. 3 *a* and *b*). On roentgenoscopic examination reduction in the amplitude of cardiac contractions was noticed. Preoperative treatment for elimination of fluids from body tissues was instituted.

Operation.—May 9, 1938: Marked thickening of the pericardium and epicardium was found together, with extensive deposits of calcium on and between these layers. The scar was adherent over the entire myocardium. Extensive epicardiolysis was performed over both ventricles and the right auricle to the vena cava orifice, with resection of a large portion of the pericardial and epicardial scar from the anterior and lateral surfaces of the heart (Fig. 4*a*). Cultures of the pericardial scar were negative and microscopic examination of it revealed inflammatory tissue and calcium, without any evidence of tuberculosis.

The immediate convalescence was satisfactory. Influenza developed two weeks after the operation which delayed the convalescence somewhat. At the time of dismissal two and one-half months after operation, the ascites had almost disappeared (Fig. 4*b*). Tests of liver function revealed retention of dye Grade 2, whereas before operation it was Grade 3. The venous pressure was 156 Mm. of water, whereas, before operation

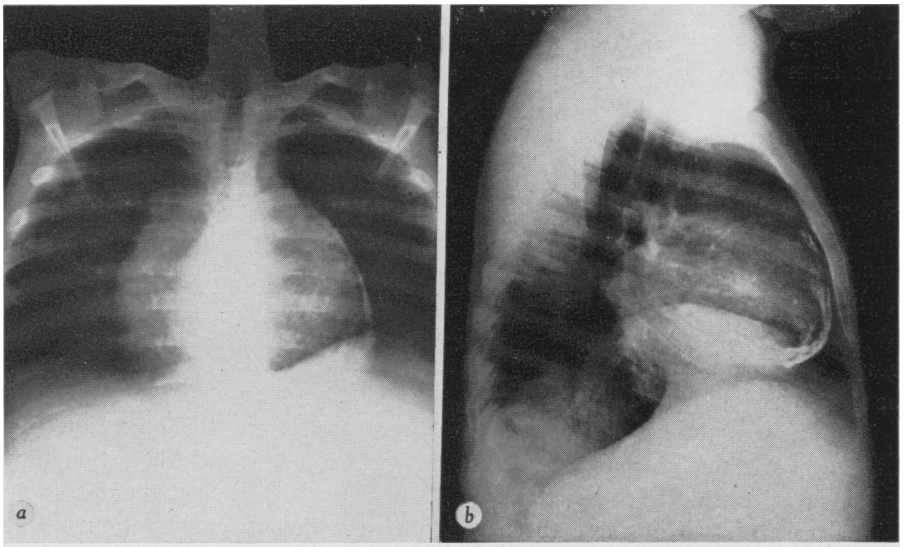

FIG. 3.—Enlargement of the heart shadow and deposits of calcium at time of admission; *a*, antero-posterior and *b*, lateral views.

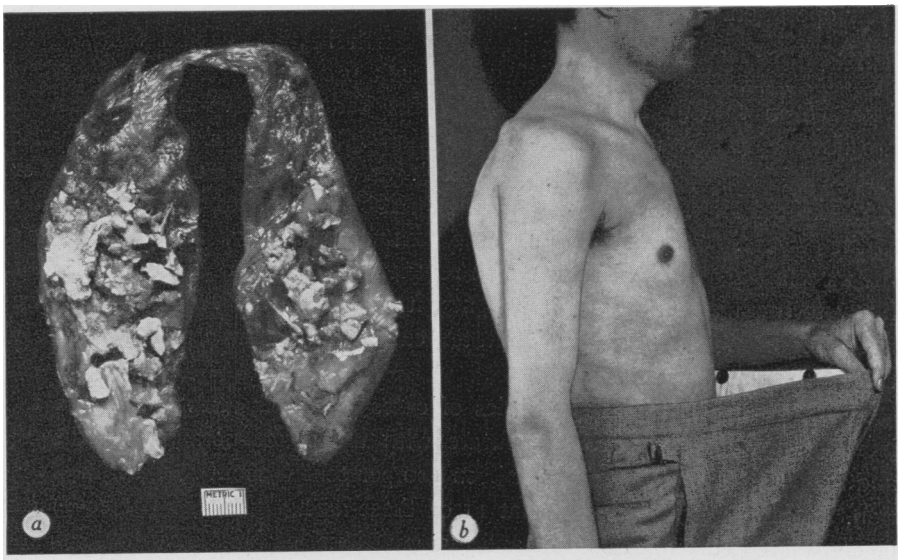

FIG. 4*a*.—Typical pericardial scar and calcium removed at operation together with the cartilages resected from the wall of the thorax. *b* (Case 1): Marked reduction in the size of the abdomen after operation owing to relief of ascites is indicated by size of waistband of trousers worn by patient on admission.

it was 310 Mm. The circulation time was 20 seconds, whereas, before operation it was 43 seconds. The patient gradually improved for nearly two years. At the end of that time he was considered cured. He is now leading an active and useful life.

COMMENT

The results obtained for the 18 patients who recovered from operation, I believe, are satisfactory. Nine of these patients are considered cured. The term "cured" is used in a restricted sense to designate the results of these operations. It means here the relief of subjective symptoms and the rehabilitation of the patient to his former or some useful occupation. The actual damage to the heart undoubtedly is not entirely relieved and the life expectancy of these patients is probably not as great as it could have been had they not been afflicted by the disease. The condition of two more patients has improved markedly and their improvement has approximated a cure. Operations have been done only recently in two other cases in which progressive improvement has occurred. These four patients may ultimately be considered cured, thus making a total of 13 of this series of 24 patients who ultimately may be considered as cured.

Of the remaining five patients who recovered from the operation two have shown a moderate degree of improvement but it is not likely that they will ever be considered as cured. Three patients have died since the operation. The deaths of two of these three patients were due to continuation of the disease. One was due to progressive cardiac failure and the other to tuberculous peritonitis. In the remaining case the patient was improving satisfactorily from operation but unfortunately died of pneumonia seven months after operation.

A review of the six cases in which death in the hospital followed the operation, indicates that a satisfactory result probably would not have been obtained in three. Two of these patients had extensive tuberculosis and the other extensive myocardial degeneration with calcification of the wall of the ventricle. The remaining three patients who died in the hospital were children and all were in poor general condition because of the long duration of the disease. On examination of the hearts at necropsy, marked atrophy of the muscle was found. Inasmuch, as regeneration of the myocardium is more likely to occur among younger patients, it is unfortunate that the lesion was not recognized and operation instituted early in the course of the disease in these children.

The mortality rate in this series of cases is 25 per cent. Although this is relatively high, it is commensurate with the seriousness of the disease, and the mortality rate would have been 100 per cent without surgical intervention. It is to be remembered that 11 of these 24 patients (46 per cent) can be considered cured at this time and two additional patients may subsequently improve to this degree of recovery. I believe that the percentage of cures can be increased greatly by earlier recognition of the disease and the institution of surgical treatment.

Those patients who respond to operative treatment present one of the most dramatic results obtained from surgical procedures, and it is most gratifying to see that these patients who are doomed to a slow, lingering death may be restored to health and usefulness.

REFERENCE

[1] Harrington, S. W., and Barnes, A. R.: Diagnosis and Surgical Treatment of Chronic Constrictive Pericarditis. South. Surgeon, 9, 459–484, July, 1940.

DISCUSSION.—DR. JOHN ALEXANDER, Ann Arbor, Mich.: A matter worth mentioning is the probable value of a two-stage operation for those patients who have had evidence of cardiac constriction for a year or more, and who presumably have an atrophied myocardium.

I recall that about ten years ago I extensively released both the left and right ventricles at one operation in a patient who had had cardiac constriction for a number of years. The heart dilated and the patient died seven days after operation. Since then Doctor Haight and I have operated in two stages in those few patients whose myocardial sufficiency was probably considerably impaired, freeing that part of the heart to the left of the midline at a first operation, and to the right of the midline at a second operation, if the signs and symptoms were not adequately relieved one or more months after the first operation. Most of the patients have not needed the second or right-sided stage; we have employed two stages in three patients, without a hospital death.

Incidentally, we have been surprised by the absence of severe reactions from pericardiectomy in our cases of chronic tuberculous pericarditis.

DR. CLAUDE S. BECK, Cleveland, Ohio: I do not think we should have any difficulty in the recognition of this condition. As a matter of fact, I think quite frequently you can make a diagnosis by inspection of the patient alone. You cannot always do that, but you can do it in some patients.

There is a very simple diagnostic triad for this condition which I should like to present to you, if I may.

The term which I like to apply to this condition is "compression scars of the heart" instead of "constrictive pericarditis". Here is the diagnostic triad which I think makes the diagnosis perfectly simple and infallible:

The most important features of the triad is a small, quiet heart. A compressed heart is a small atrophic organ. The muscle fibers are smaller than normal. We showed that some years ago. This type of heart is atrophic; it has undergone atrophy of disuse.

I should like to point out that the heart in this condition does not waste any energy. It is a perfectly efficient "pump". There is no valve leakage. The only thing is it does not get enough work to do. It does not receive enough blood to do the normal quota of work, and it undergoes atrophy from disuse, and that is why when you take the scar off you get an immediate improvement, and then later on you get a delayed improvement because this atrophy from disuse requires time for it to disappear. A compressed heart cannot dilate nor can it hypertrophy.

The fluoroscope usually helps you to recognize a quiet heart, but the shadow you see in the roentgenogram or in the fluoroscope is not always just heart—it is heart plus the compression agent, and the compression agent may be in some cases fluid, blood or pus. It may be a tumor, and in other cases it may be quite a thick scar.

As Doctor Harrington has shown, the scar may be one centimeter or more in thickness. The shadow may appear to be large, whereas, the heart actually is a small organ.

The other two components of this triad refer to the superior and inferior vena cava. These structures always contain blood under pressure. When the venous pressure rises to a level of 18 or 20 cm. then the fluid part of the blood begins to filter through the capillary bed, and you get ascites. It is ascites that takes the patient to the doctor.